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General Medicine

A CASE REPORT ON NEUROTOXIC SNAKE BITE

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ABSTRACT In the Indian subcontinent, snakebite is a highly prevalent tropical disease that is often ignored. The amount of venom injected, its potency, the type of snake, how long the patient was hospitalized, and when and how much anti-snake venom (ASV) was administered all affect how severe the respiratory muscle paralysis is and how long it takes to recover. There is yet no clear explanation for his delayed neuromuscular recovery. We describe one such example of a young adult who, after a neurotoxic snakebite, required prolonged ventilatory support and had delayed neuromuscular recovery.

KEYWORDS : Neurotoxicity, Respiratory Failure, Delayed Recovery, Mechanical Ventilation, Krait, And Snakebite

INTRODUCTION

Envenomation from a snakebite is referred to as a "neglected tropical disease" by the World Health Organization [1]. In Southeast Asia and the Indian subcontinent, snakebite is a common occurrence. Since many patients choose not to report their snakebites to the authorities, the disease's mortality and morbidity have historically been underestimated [2, 3]. According to data from the Million Death Study, envenomation from snakebite could account for up to 45,900 deaths every year. Additionally, it was discovered that the monsoon season had the highest frequency of these deaths and that the majority of deaths occur in rural India where there is little access to basic healthcare [4].

The patient's clinical signs determine how neurotoxic envenomation should be managed. The use of polyvalent anti-snake venom (ASV) may help recover the basic neurotoxic symptoms of muscle paralysis. Additional ventilatory support may be required due to the involvement of the bulbar and respiratory muscles [5]. Recovery times also differ; some patients recover in 24 to 48 hours after therapy, while others need continuous ventilator assistance. Additionally, some individuals may experience a delayed onset of neurological symptoms that resembles critical illness polyneuropathy (CIP) and Guillain-Barré syndrome (GBS), necessitating a lengthier recovery period [6–9]. Here, we describe the case of an 32-year-old guy who had a hospital stay and displayed symptoms of a neurotoxic snakebite.

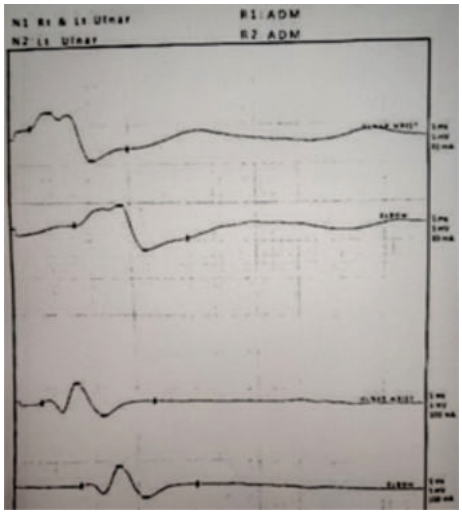

Figure 1: Nerve conduction study of the ulnar nerve showing increased sensory latency and reduced SNAPs

Figure2: Ptosis and Diplopia shown by patient

Case Study

An 32-year-old young male, a construction worker by occupation, was brought to the emergency department by his friends around 4 am; he has a history of snakebite (common krait) on his right hand around 2 am while he was sleeping. The patient subsequently developed swelling of his right hand and forearm, followed by multiple episodes of vomiting. He became drowsy subsequently. He was brought to the hospital after receiving first aid at a nearby clinic. At the time of admission, the fang mark was noted on the dorsal aspect of the right hand near the base of the thumb. He was drowsy (Glasgow Coma Scale: 8/15), tachycardic (124 beats/minute), tachypneic (32 breaths/minute), hypertensive (180/110 mm Hg), and hypoxemic (SpO_2: 60% on room air, PaO_2: 54 with 15 L of O_2) with severe respiratory acidosis (pH: 7.05, pCO_2: 84 mm Hg). Neurological examination revealed flaccid quadriparesis with bilateral ptosis and bilateral external ophthalmoplegia with pupils sluggishly reacting to light. Other system examinations were unremarkable. In view of his worsening respiratory failure, he was emergently intubated and started on mechanical ventilation.

His initial blood tests came back within normal bounds. His complete

blood clotting time of twenty minutes showed no anomalies related to coagulation. After receiving 10 vials of polyvalent ASV intravenously over the course of 30 minutes in normal saline, his neurological condition did not improve. Therefore, 10 further vials of polyvalent ASV were administered in a similar manner over a further 30 minutes. The patient was now sent to the intensive care unit in order to receive additional care. After a thorough neurological evaluation, it was discovered that the patient had total ophthalmoplegia and dilated pupils. There was no reaction to painful stimuli, and there were no deep tendon responses.

On day 7, a nerve conduction examination was performed since he was still experiencing a chronic neuromuscular weakness (power: grade 3 in all four limbs, reduced deep tendon reflexes). An increase in the bilateral median and ulnar nerve's sensory delay was observed, along with bilateral sensory and motor axonal forms of polyneuropathy. Additionally, there was a decrease in the bilateral median and superficial peroneal nerve sensory nerve action potentials (SNAPs) (Figure 1)

He was eventually taken off the ventilator on day 10 of his hospital stay, and he was breathing room air while maintaining a sufficient saturation level. He was still receiving physical therapy and his muscle weakness persisted. On day 12, he was moved out of the ICU and received care in the ward. He was allowed to leave the hospital after showing improvement in his power (grade 4+ in all four limbs). It was suggested that he get regular follow-up.

DISCUSSION

The Elapidae family, which includes the cobra and krait species, is home to the majority of the venomous snakes found in India [5]. These snakes can cause neurotoxicity when they poison. Neurotoxicity can cause direct toxicity to the nerves as well as abrupt neuromuscular paralysis of all muscles, including the bulbar and respiratory muscles. Seasonal, regional, and genetic differences within the species changes in the venom's constituent toxins have all been linked to differences in the clinical presentations of patients suffering from neurotoxic envenomation. Many factors could be at play in individuals whose snakebite symptoms take a long time to appear, including the amount of venom injected, the type of venom injected, the delay in presenting to the hospital the dose of the ASV administered [10]

Neurotoxins categorized as alpha and beta bungarotoxins are found in Elapidae venom. Whereas beta toxins function presynaptically, alpha bungarotoxins function postsynaptically [11]. The primary component of beta bungarotoxins is phospholipase A2, which can cause the skeletal muscle's motor nerve terminals to lose synaptic vesicles. This can then cause the motor nerve terminal to be destroyed and the intramuscular axons' cytoskeleton to degenerate because it disturbs the phospholipids found in the cell membranes [12]. This may help to explain the pattern of axonopathy observed in our patient's nerve conduction investigation. The length of a patient's respiratory paralysis can vary, as the loss of the motor terminal is typically irreversible and the patient's clinical recovery depends on the regeneration of new nerve terminals [13]. A direct systemic effect of the envenomation was attributed to the considerable abnormalities, including reduction in conduction velocities and extension of sensory, motor, and f-wave latencies, that were observed in patients with delayed recovery, according to the literature [14].

Polyvalent ASV is typically used to counteract the circulating venom. The toxin that has already attached itself is not neutralized and can still work. This may be the cause of each patient's inability to react appropriately to the highest dosage of ASV. In this sense, the results of patients suffering from snakebite envenomation may differ depending on the prompt and appropriate delivery of ASV [14]. Acetylcholinesterase inhibitors are exclusively effective against postsynaptic toxins; they are ineffective against presynaptic toxins in reversing the neuromuscular paralysis caused by snake envenomation [10].

CONCLUSIONS

Our research leads us to the conclusion that the mainstay of treatment for neurotoxic envenomation with respiratory failure is the administration of anti-snake venom combined with anticholinesterase medication and cardio-respiratory support. If treatment is initiated early and prior to an irreparable hypoxic injury, the outcome is favorable.

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